V F. Blood Cells and Plasma

Exposure to carbon monoxide causes an increase in platelet adhesiveness in atherosclerotic patients (El-Ebrashy et al, 1967) and in rabbits (Birnstingl et al, 1971). There is also an increase in fibrinolytic activity in patients suffering from carbon monoxide poisoning (El-Attar, 1968 a and b) and in experimental animals (Candura and Craveri, 1964). However, none of these effects have been encountered as a result of cigarette smoking.

The concentration of enzymes in the blood reflecting cardiac and liver damage have been reported in patients suffering from acute carbon monoxide poisoning (Ricci et al, 1964; Van Vugt, 1968; Duplay et al, 1967; Afans'ev, 1967; Gramer and Ruof, 1968; Müller and Voigt, 1968; Prellwitz et al, 1970; Antos and Sevcik, 1971).

Four groups of situations are characterized by a similar hematologic response. They are as follows:

1. Exposure to carbon monoxide in man causes an increase in the red cell and reticulocyte counts and the serum globulin fraction (Coscia et al, 1964; Tkachenko et al, 1966; Glass et al, 1968; Kjeldsen and Damgaard, 1968; Bethlenfalvay, 1971a). The erythrocytes manifest some differences from normal erythrocytes in staining (Blackmore, 1970; Bethlenfalvay, 1971b), denaturation of hemoglobin (Perrelli et al, 1970) and a change in the shape of the oxyhemoglobin dissociation curve to the left (Brody and Coburn, 1969).

2. In dogs, exposure to carbon monoxide increases hemoglobin

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concentration (Asmussen and Vinther-Paulsen, 1949). This change is regarded as an important mechanism for tolerance or acclimatization to carbon monoxide (Otis, 1970). A similar increase in hemoglobin and the red cell count has been noted in other species, such as rabbits (Truhaut et al., 1968), rats (Ramsey, 1969), rhesus monkeys (Theodore et al., 1971), and squirrel monkeys (Jones et al., 1971).

3. Cigarette smoking causes an increase in the red cell count and hemoglobin, whereas abstinence caused a fall in the content of these in humans (Eisen and Hammond, 1956; Pincherle and Shanks, 1967) and in hamsters (Reckzeh and Dontenwill, 1970). In another investigation involving smokers, the elevated hemoglobin and red cell count were correlated with carboxyhemoglobin levels (Petrovic, 1970). A shift in the oxygen dissociation curve to the left has been noted in cigarette smokers (Gutenkauf et al. 1967; Birnstingl et al., 1967).

4. Patients suffering from myocardial ischemia with normal coronary arteriograms have been shown to have abnormal hemoglobin-oxygen dissociation (Likoff et al., 1967; Eliot and Bratt, 1969; Guy et al., 1971). There is no evidence that these patients sustain carboxyhemoglobinemia, nor is the ischemia associated with cigarette smoking.

The above examples are clearly different entities. Cigarette smoking is included only in situation 3 and carbon monoxide in 1 to 3.

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VI. NERVOUS SYSTEM

Death from carbon monoxide poisoning is caused by direct depression of the centers in the medulla oblongata. The other parts of the central nervous system are influenced by carbon monoxide. The reports on the clinical and histopathological manifestations of disturbances of the central nervous system are listed in additional bibliography no. 11. This section will review selected topics relating to the effects of sublethal concentrations of carbon monoxide on the nervous system and its related structures.

A. Cerebral Blood Vessels

The following cerebral lesions resulting from acute carbon monoxide poisoning have been reported: cerebral hemorrhages (Breitenecker, 1938; Wajgt, 1971); obstruction of basilar artery (Breton et al., 1969); cerebral vascular thrombosis (Glowacki et al., 1958; Saita and Lussana, 1971). In anesthetized cats the administration of 0.2 to 0.5% carbon monoxide caused an elevation of intracranial pressure accompanied by dilatation of pial vessels and an increase in cerebral blood flow (Maurer, 1941; Sjostrand, 1948; Fodor, 1969; Hall 1972). The cerebral vessels, like other vessels, dilate during carboxyhemoglobinemia. The increase in caliber of the vessels leads to hemorrhage and thromboses.

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VI B. Eye and Visual Pathways

Visual threshold is influenced by administration of carbon monoxide (see review by McFarland, 1970). McFarland et al. (1944) used a visual discriminometer and noted a change in visual sensitivity following exposure to 0.01% carbon monoxide. There was a fall in threshold with a rise of 5% in carboxyhemoglobin level in 2 subjects. Lilienthal and Fugitt (1946) noted in 5 subjects that increments of 5 to 10% carboxyhemoglobin impaired the critical flicker fusion frequency. Halperin et al. (1959) observed the recovery from the effects of carbon monoxide on visual function. Administration of pure oxygen resulted in an immediate improvement, and the addition of 7% carbon dioxide to the oxygen hastened the elimination of carbon monoxide from the body, compared with the inhalation of air.

In patients suffering from carbon monoxide poisoning the following effects have been reported: change in optically evoked response in the electroencephalogram (Helmchen and Künkel, 1964; Hosko, 1970); impairment of visual perception relating to lesions in the occipital lobe (Kuroiwa et al., 1967; Szliwowski and Klees-Delange, 1970), visual agnosia (Zolotukhin, 1968; Benson and Greenberg, 1969), retinopathy (Heydenreich, 1970; Bilchik et al., 1971) and conjunctival hemorrhages (Prokop and Wabnitz, 1970).

In laboratory animals inhalation of carbon monoxide leads to lesions which relate to those observed in human poisoning. In rabbits there is sludging of blood in the ophthalmic vessels (Taccola et al., 1965). Diminution of evoked potentials in the visual pathways occurs in cats (Ikeda, 1969) and in

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rabbits (Katsura, 1971). Cytochemical studies in the guinea pig (Niebr6j, 1969) and electron microscopic observations in the mouse (Ohara, 1968) indicate some degenerative changes in the retina.

The carbon monoxide contained in cigarette smoke has not been compared with carbon monoxide administered as a gas mixture. The effect of cigarette smoking on vision has been tested by Melanowski (1963) and Schmidt (1970). The techniques applied to smoking are different from those reported above for the investigation of carbon monoxide, so that it is not possible to assess the influence of carbon monoxide contained in cigarette smoke on vision.

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VI. NERVOUS SYSTEM

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VI C. Hearing and Auditory Pathways

The auditory and vestibular apparatus is less sensitive than the eye to carboxyhemoglobin. Tibbling (1969) tested 25 human volunteers by the smoking of cigarettes. A rise in the blood level of carboxyhemoglobin to 2.5% did not influence vestibular nystagmus. Guest et al. (1970) administered carbon monoxide to produce levels of 10% carboxyhemoglobin in 8 subjects. No depression of the auditory flutter fusion threshold or of the critical flicker fusion threshold occurred. In the same subjects the oral administration of phenobarbital depressed both thresholds.

Patients suffering from acute carbon monoxide poisoning develop the following disorders relating to the ear: abnormalities in hearing (Taniewski and Kugler, 1964a; Hansz and Styperek, 1968; Kawamura, 1971); deafness (Morris, 1969; Fortunato and Catalano, 1970); distortion of the audiometric curve (Taniewski and Kugler, 1964b; Fritsche, 1969); and disturbance in cochleovestibular function (Cis and Perani, 1964; Mounier-Kuhn et al., 1968). Industrial workers exposed chronically to carbon monoxide develop similar abnormalities in hearing and vestibular function (Stanković et al., 1964; Strzelczyk and Zenk, 1964; Zenk, 1964; 1965; Sato, 1966; Mesolella et al., 1970.

In experimental animals the administration of carbon monoxide causes interference with the auditory and olfactory systems. There is depression of action potentials in the olfactory bulb of the cat (Hall, 1970) and in the cochlea of the guinea pig (Freigang et al., 1968). The morphologic examination of the inner ear shows degeneration in the rabbit (Küttner, 1968) and in other animal species (Kittel and Theissing-Erlangen).

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VI. NERVOUS SYSTEM

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VI D. Behavior of Man

The effects of carbon monoxide on human behavior have been summarized by McFarland (1952), Beard and Grandstaff (1970) and LaVerne (1970). Several reports have appeared which determine the blood level of carboxyhemoglobin that would influence functions of the higher centers of the central nervous system in man. The results are as follows:

1. Sayers et al, (1929) summarized the findings of an investigation in which 6 men were exposed for 4 to 7 hours daily over a period of 68 days to gasoline engine exhaust gas-air mixtures. Exposure to 200 ppm carbon monoxide, resulting in blood levels of 20% carboxyhemoglobin, caused frontal headache in 3-1/2 to 4 hours. Exposure to 300 ppm, producing a blood level of 30%, caused frontal and occipital headaches and vertigo after 3 hours. Nielsen (1971) in another group of subjects with 25% to 33% carboxyhemoglobin noted an elevation of body temperature.

2. The control precision and multiple limb coordination are impaired when the concentration of carboxyhemoglobin in the blood exceeds 5% (Trouton and Eysenck, 1961).

3. Psychomotor abilities are sensitive to the presence of carboxyhemoglobin. Ability to discriminate is altered at levels below 5%, while reaction time, static steadiness and muscle persistence are measurably altered by concentrations of up to 20% (Schulte, 1963). There was no difference between the test results in nonsmokers and those in smokers.

4. The ability of subjects to estimate time intervals of 1, 3 and 5 seconds was not impaired by exposures to as much as 20% carboxyhemoglobin.

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Levels between 20 and 30% caused interference with visual evoked response and impairment of manual dexterity (Stewart et al., 1970).

5. Estimation of time and tracking performance was not influenced by exposure to 125 ppm carbon monoxide with a mean of 6.64% carboxyhemoglobin (Hanks, 1970; Mikulka et al., 1970; O'Donnell et al., 1970, 1971 a and b).

6. Levels of carboxyhemoglobin below 10% influence the results of tests for automobile driving performance (Ray and Rockwell, 1970).

7. Levels of 6.6% carboxyhemoglobin caused a reduction in the ability of the subjects to identify signals. This effect on vigilance did not appear with 2.3% carboxyhemoglobin (Horvath et al., 1971).

8. Exposure of students to 100 ppm carbon monoxide for 2½ hr, with mean levels of 7.2% carboxyhemoglobin, resulted in diminution of manual dexterity, visual perception and ability to learn and perform certain intellectual tasks (Bender et al., 1971). There was no discernible difference between smokers and nonsmokers with regard to their psychological susceptibility.

9. Ramsey (1972) exposed 20 normal subjects and 20 patients with pulmonary emphysema and 20 with anemia to inhalation of 0.03% carbon monoxide in air for 40 min. The mean increase of 4.5% in carboxyhemoglobin caused a diminution in the speed of reaction to a visual stimulus but no significant change in tests for depth perception and for visual discrimination of brightness.

10. The only observations relating to cigarette smoking pertain to high altitude. Kratochvil et al. (1957) tested smoking at a level of 18,000 feet. There was only a slight decrement in performance of subjects when smoking while hypoxic at high altitude compared with that of the hypoxic controls. Astrup et al. (1971)

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reported on a patient who suffered from mountain sickness over many years. The attacks were provoked either by cigarette smoking or by inhalation of 0.5% carbon monoxide. There are no other reports of such patients with mountain sickness induced by tobacco smoking. It is possible that these responses may relate to changes in cerebral blood flow described by Demange and Auzas (1969).

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VI. NERVOUS SYSTEM

D. Behavior of Man

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VI. E. Behavior of Animals

The effects of carbon monoxide on the behavior of certain animals have been described, viz, on that of rats (Beard and Wertheim, 1967; Goldberg and Chappell, 1967; Teichner, 1967; Stupfel et al, 1968; Khachaturiyan et al, 1969; Rose et al, 1970; Stupfel and Bouley, 1970; Nečas and Neuwirt, 1971), mice (Hirata et al, 1969; Gaume et al, 1971), dogs (Carding, 1968; Preziosi et al, 1970), monkeys (Back, 1969), and laboratory animals (Fazekas, 1967). The morphologic and functional features of the brain have been described, resulting from exposure of various animals to carbon monoxide, viz, rats (Thomas and Pearse, 1964; Miyagishi and Hiyashi, 1968; Miyagishi and Suwa, 1969; Takahata and Miyagishi, 1969), mice (Estler et al, 1971A), guinea pigs (Kupfer and Wünscher, 1968), cats (Ando et al, 1969; Barrios et al, 1969), birds (Dugnat, 1965), and frogs (Segal, 1970). The brain exposed to carbon monoxide shows a reduction in content of gangliosides (Mawatari, 1970), accumulation of ammonia (Okunyev and Prokhorenko, 1965; Mishchenko and Frenkel, 1966), decrease in catecholamines (Marks and Swiecicki, 1971); decrease in serotonin (Pare et al, 1969), and decrease in glycogen (Estler et al, 1969, 1971b). The relationship of these results to the behavior pattern has not been elucidated.

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